

Madison, Wis.
December 1, 1955

Dear Francois:

[Saer]

As I should have done sooner, I am enclosing some reprint copies which arrived recently of a letter to Science which embodies some of the points raised in our earlier correspondence. Actually, I was impelled to write this comment primarily by Mark Adam's newsreport which, I felt, went too far in insisting on the finality of a scientific issue which is obviously open to further revision. I am sure that my position will be misunderstood elsewhere, but I hope that you will understand that I am merely asking for an openminded outlook until experiments which are more decisive than haploid segregation can be devised.

Meanwhile, I am astonished to learn that some of the discrepancies in our results may be based on different behavior of Hfr strains-- at least this seems to be the conclusion, or supposition, of Garen and Skaar. I have not made a direct comparison myself; I trust you have the M- Hfr cavalli stock; if not, please let me know, or get it from Luca, as it has been freely distributed for quite a long time now. This discrepancy, if there is one, may explain many things I could not understand before, e.g., your reported frequency of syngamic induction, Gal ratios, etc., which we have not seen in our crosses.

I hope you will give special consideration to the hypothesis in the enclosure that perhaps the Hfr chromosome is generally already broken in the gamete, but that the terminal fragment may or may not get into the F- cell. This would be quite consistent with the diploid experiments: i.e., the "type A" diploids would come partly from pre-, partly from postzygotic losses of the Mal6S segment, but they constitute at most 85% of the total, and the presygotic loss may have any smaller value. This event may be what is affected by blending. At any rate, I still wish there were explicit evidence, from diploids, of variability in the location of breaks; so far there is none. It was especially interesting that you brought up the segregation in transduction clones. I am rather confused by the status of this phenomenon in Lennor's system. With lambda, the "heterogenotes" are a regular event, and their orderly segregation and cross-over behavior has greatly illuminated our understanding of transduction. This work, which Dr. Morse is primarily responsible for is being prepared for publication/and should be out soon in Genetics (at least the first installment).

Are you going on with your transduction studies, with other markers? There are some questions on the genetics of the Lac loci that I would like to reanalyse more fully by transduction analysis, perhaps sometime next year, for which the comparative properties of different phage systems would be most useful, but I do not wish to intrude on problems in which you may have an immediate interest. At the moment, I am mainly preoccupied with developing stocks to permit diploid analysis to be conducted on a more routine basis (e.g., by crosses of diploid x haploid) but this is proving to be an extremely tedious, time-consuming and tedious task, which has given very little fruit even since early summer. I might ask you to tell Jacques that our Het stocks are surely less potent than formerly, and we have so far gotten only one diploid (not a satisfactory one for our purposes) from crosses with constitutive. We are continuing the search.

I understand you may come to the States next summer for McElroy's symposium. I do not know what our travel funds will be then, but I hope your plans will include enough time for the possibility of a visit with us. Sincerely,

John D. Doe